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The role of open abdomen in non-trauma patient: WSES Consensus Paper

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Abstract

The open abdomen (OA) is defined as intentional decision to leave the fascial edges of the abdomen un-approximated after laparotomy (laparostomy). The abdominal contents are potentially exposed and therefore must be protected with a temporary coverage, which is referred to as temporal abdominal closure (TAC). OA use remains widely debated with many specific details deserving detailed assessment and clarification. To date, in patients with intra-abdominal emergencies, the OA has not been formally endorsed for routine utilization; although, utilization is seemingly increasing. Therefore, the World Society of Emergency Surgery (WSES), Abdominal Compartment Society (WSACS) and the Donegal Research Academy united a worldwide group of experts in an international consensus conference to review and thereafter propose the basis for evidence-directed utilization of OA management in non-trauma emergency surgery and critically ill patients. In addition to utilization recommendations, questions with insufficient evidence urgently requiring future study were identified.

Keywords: Open abdomen, Laparostomy, Non-trauma, Peritonitis, Pancreatitis, Vascular emergencies, Fistula, Nutrition, Re-exploration, Re-intervention, Closure, Biological, Synthetic, Mesh, Technique, Timing

Background

The decision by a surgeon to utilize the open abdomen (OA) technique is a dramatically non-anatomic situation that dramatically increases resource utilization and has potential severe side effects. It is, however, often dramatically effective at countering the drastically impaired physiology of critical illness when no other perceived options exist. There are both mandatory and relative indications for OA use, which are heavily influenced by the primary pathophysiologic insults and responses to intra-abdominal sepsis and inflammation, both inherent to the patient and induced through medical treatments. The abdominal compartment is dramatically affected in both its contents and the characteristics of the abdominal wall. Several factors as systemic inflammatory response syndrome, increased vascular permeability, and aggressive crystalloid resuscitation predispose to fluid sequestration leading to peritoneal fluid formation. Patients with severe sepsis and septic shock commonly receive large amounts of resuscitation fluids and may develop excessive gut edema and diminished contractility and motility. These changes in combination with sequestration of second and third space fluids and forced closure of an abdominal wall with altered compliance may result in increased intra-abdominal pressure (IAP) ultimately leading to intra-abdominal hypertension (IAH) or even abdominal compartment syndrome (ACS) [1, 2].

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The pathophysiologic implications of elevated IAP have been restarted to be studied in deep during the last 20 years [2–4]. In 2013, The Abdominal Compartment Society (WSACS) updated the previously published definition and guidelines for the management of intra-abdominal hypertension [5]. Elevated IAP constitutes IAH and was classified into four grades: (1) grade I IAP 12–15 mmHg, (2) grade II IAP 16–20 mmHg, (3) grade III IAP 21–25 mmHg, and (4) grade IV IAP >25 mmHg. Elevated IAP commonly causes marked deficits in loco-regional and whole body perfusion that may result in organ failure [5]. An uncontrolled IAH, with an IAP exceeding 20 mmHg and new onset organ failure, is defined as an abdominal compartment syndrome (ACS) [2, 5]. ACS is a syndrome and not a disease, as such, it can have many causes and it can occur in many disease processes, it is an all or nothing phenomenon, while IAH is a more graded continuum. ACS in turn has further effects on intra-abdominal organs, as well as indirect effects on the other organ(s) and system(s). The ACS is a potentially and frequently lethal complication characterized by effects on splanchnic, cardiovascular, pulmonary, renal, and central nervous systems [2, 5]. While medical therapies should be attempted, the ACS is rapidly lethal and opening of the abdominal cavity conducted promptly if medical interventions do not quickly alleviate or temporize the situation. If surgery has been undertaken for the index disease, leaving the abdomen temporarily open is often required to prevent inducing ACS in a critically ill pro-inflammatory patient with visceral edema and ongoing resuscitation. Whether leaving the abdomen open will primarily influence the septic response is also intriguing but unproven at the present time.

The OA procedure is defined as intentionally leaving the fascial edges of the abdomen un-approximated (laparostomy). The abdominal contents are exposed and thus must be protected with a temporary coverage, which is itself termed a temporary abdominal coverage (TAC) [2, 6]. The OA technique, when used appropriately, may be useful in the management of surgical patients with compromised general conditions due to any critical illness/injury but most frequently cases of intra-abdominal sepsis and severe pancreatitis are seen recently [7]. Despite many serious potential complications, the OA is perceived to be a life-saving intervention in catastrophically injured patients [2]. Compared to trauma patients, however, patients undergoing OA management for intra-abdominal non-trauma emergencies have greater risks subsequent to OA utilization, including entero-atmospheric fistula (EAF) and a "frozen abdomen", intra-abdominal abscesses, and lower rates of definitive fascial closure [8, 9] with resultant large ventral hernia defects. This discrepancy in risks and benefits, along with economic considerations [10], was the primary reason the WSACS suggested not routinely using the OA for septic cases versus traumatic cases [5]. Thus, every effort should be exerted to attempt abdominal closure as soon as the patient can physiologically tolerate it.

Methods

The recommendations are formulated and graded according to the *modified Grading of Recommendations Assessment, Development and Evaluation* (GRADE) hierarchy of evidence from the GRADE Group, summarized in the Table 1 [11].

The WSES and Abdominal Compartment Society together with the Donegal Research Academy united a group of subject-matter experts coordinated by a central coordinator to review and summarize the evidence and thereafter to express their evidence-based opinion on important issues concerning OA utilization in non-trauma patients:

Which non-trauma patients can benefit from OA techniques and for which specific critical conditions is indicated (example, peritonitis, vascular emergencies, and severe pancreatitis)?

What is the optimum TAC technique for use in non-trauma patients?

Is there a role for fluid instillation?

What is the optimum timing of re-exploration before definitive closure in non-trauma patients?

What is the optimum timing to definitively close an OA in non-trauma patients?

What are the optimum adjunctive techniques to definitively close an OA in non-trauma patients considering both non-mesh-mediated techniques and mesh-mediated techniques?

What is the optimum treatment to treat frozen abdomen and enteral fistulas?

What nutritional support is indicated in OA?

A central project coordinator compiled the answers and statements derived from the first round of presentations and discussions. The statements were discussed during the Consensus Conference held in Dublin (Ireland) in July 2016. Once an agreement was reached within the experts groups, a final round of discussion among a larger group of experts led to the final version of recommendations reflecting the final expert-consensus document (Table 2).

Open abdomen in peritonitis

The open abdomen is an option for emergency surgery patients with severe peritonitis and septic shock under the following circumstances: abbreviated laparotomy due to the severe physiological derangement, or the need for a deferred intestinal anastomosis or a planned second look

Table 1 "Modified Grading of Recommendations Assessment, Development and Evaluation (GRADE)" hierarchy of evidence from the American College of Chest Physicians task force by Guyatt and colleagues [11]

Grade of recommendation	Clarity of risk/benefit	Quality of supporting evidence	Implications
1A			
Strong recommendation, highquality evidence	Benefits clearly outweigh risk and burdens, or vice versa	RCTs without important limitations or overwhelming evidence from observational studies	Strong recommendation, applies to most patients in most circumstances without reservation
1B			
Strong recommendation, moderate-quality evidence	Benefits clearly outweigh risk and burdens, or vice versa	RCTs with important limitations (inconsistent results, methodological flaws, indirect analyses or imprecise conclusions) or exceptionally strong evidence from observational studies	Strong recommendation, applies to most patients in most circumstances without reservation
1C			
Strong recommendation, lowquality or very lowquality evidence	Benefits clearly outweigh risk and burdens, or vice versa	Observational studies or case series	Strong recommendation but subject to change when higher quality evidence becomes available
2A			
Weak recommendation, high-quality evidence	Benefits closely balanced with risks and burden	RCTs without important limitations or overwhelming evidence from observational studies	Weak recommendation, best action may differ depending on the patient, treatment circumstances, or social values
2B			
Weak recommendation, moderate-quality evidence	Benefits closely balanced with risks and burden	RCTs with important limitations (inconsistent results, methodological flaws, indirect or imprecise) or exceptionally strong evidence from observational studies	Weak recommendation, best action may differ depending on the patient, treatment circumstances, or social values
2C			
Weak recommendation, Low-quality or very lowquality evidence	Uncertainty in the estimates of benefits, risks, and burden; benefits, risk, and burden may be closely balanced	Observational studies or case series	Very weak recommendation; alternative treatments may be equally reasonable and merit consideration

for intestinal ischemia, *or persistent source of peritonitis (failure of source control), or extensive visceral edema with the concern for development of abdominal compartment syndrome* (*grade 2C*).

In severe secondary peritonitis, some patients may experience a disease progression to severe sepsis and septic shock experiencing progressive organ dysfunction, hypotension, myocardial depression, and coagulopathy and a staged approach may be required [12]. These are often hemodynamically unstable and unfit for immediate complex surgical interventions [12]. If the patient is not in a condition to be undergone to a definitive repair and/or abdominal wall closure, the intervention should be abbreviated due to suboptimal local conditions for healing and global susceptibility to spiraling organ failure. For instance, intestinal continuity restoration can be deferred to a subsequent surgical intervention, which is particularly important in hypotensive patients who are receiving inotropes [13]. In facing the impossibility to completely obtain a source control of the contamination in a single operation or if extensive visceral edema and decreased abdominal wall compliance increases the risk of ACS development, primary fascial closure should not be attempted and the abdomen should be left open [14]. The rationale for using the OA is to leave the abdomen open and to treat the infected peritoneal cavity like an "open abscess" with subsequent re-operations involving generous irrigations and potentially active TAC techniques [15] to definitively control the contamination while also preventing IAH progression to ACS. No definitive data exist about the management of severe peritonitis with the open abdomen. Robledo et al. compared open versus closed abdomen procedures in 40 patients with severe secondary peritonitis; no significant differences in mortality rates were found (55% open vs. 30% closed). The study was interrupted at the first interim analysis for high relative risk and odds ratios for death in the open group (1.83 and 2.85, respectively) [16]. However, the TAC technique that was selected as the "intervention" would be relatively contraindicated in current OA management. Some other

Table 2 Statement Grid

	Statements
Open Abdomen indication:	
➢ Peritonitis	The open abdomen is an option for emergency surgery patients with severe peritonitis and septic shock under the following circumstances: abbreviated laparotomy due to the severe physiological derangement, or the need for a deferred intestinal anastomosis or a planned second look for intestinal ischemia, or persistent source of peritonitis (failure of source control), or extensive visceral edema with the concern for development of abdominal compartment syndrome (Grade 2C).
➢ Vascular Emergencies	The open abdomen should be strongly considered following management of hemorrhagic vascular catastrophes such as ruptured abdominal aortic aneurysm (Grade 1C) The open abdomen should be considered following surgical management of acute mesenteric ischemic insults (Grade 2C).
➢ Pancreatitis	In patients with severe acute pancreatitis unresponsive to step-up conservative management surgical decompression and leaving the abdomen open is effective in treating abdominal compartment syndrome (Grade 2C) Leaving the abdomen open after surgical necrosectomy for infected pancreatic necrosis is not recommended excepted in those situation at high risk of abdominal compartment syndrome (Grade 1C)
Optimal technique for temporary abdominal closure	Negative pressure wound therapy with continuous fascial traction is suggested as the preferred technique for temporary abdominal closure (Grade 1B). Temporary Abdominal Closure without Negative pressure wound therapy (e.g., mesh alone, Bogota bag) whenever possible should NOT be applied for the purpose of temporary abdominal closure, because of low delayed fascial closure rate and being accompanied by a significant intestinal fistula rate (Grade 1B).
Is there a role for NPWT with Fluid Instillation?	There is inadequate evidence to make a recommendation regarding use of negative pressure wound therapy in combination with fluid instillation in patients with temporary abdominal closure (NOT GRADED).
Planning re-exploration before definitive closure	- In critically ill non-trauma patients with open abdomen, once any requirements for on-going resuscitation have ameliorated, early re-operation with the intention of closing the abdomen should be given a high priority (Grade 1C). - In critically ill patients with open abdomen, re-laparotomy with concern for ongoing ischemia/ contamination reoperation should be conducted no later than 24–48 h after the index operation, with the duration from the index operation shortening with increasing degrees of patient non-improvement and hemodynamic instability (Grade 1C).
Best timing to definitively close an open abdomen	- Fascia should be closed as soon as possible (Grade 1C). - Acidosis (pH <7.25), hypothermia (temperature < 34 °C) and coagulopathy (TEG, INR) are not predictive of the need for maintaining the open abdomen in non-trauma patients (Grade 2A). - The abdomen should be maintained open in non-trauma patients if the source of contamination persists, if a condition of haemodynamic instability persists meaning in presence of on-going fluid resuscitation or vasopressor support necessity, if a deferred intestinal anastomosis is needed, if there is the necessity for a planned second look for ischemic intestine and lastly if there are concerns about abdominal compartment syndrome development (Grade 2C). - Early fascia closure (within 7 days) should be the strategy for management of the open abdomen once the source control has been reached, the severe sepsis has been controlled meaning that the patient is haemodynamically stable and the hypoperfusion has been definitively corrected, no further surgical re-exploration is needed and there are no concerns for abdominal compartment syndrome (Grade 2C).
Best solution to definitively close an open abdomen	
➢ *Non-mesh mediated techniques*	- Primary fascia closure is the ideal solution to restore the abdominal closure (2A). - Component separation is an effective technique; however, it's early use is NOT recommended in fascial temporary closure. It should be considered only for definitive closure or reconstructive interventions (Grade 2C) - Planned ventral hernia (skin graft or skin closure only) remains an option for complicated open abdomen (i.e. in the presence of entero-atmospheric fistula or in cases with a protracted open abdomen due to underlying diseases) or in those low resource setting where no other facilities are present (Grade 2C)
➢ *Mesh mediated techniques*	- A fascial bridge using prosthetic mesh (polypropylene, polytetrafluoruroethylene (PTFE) and polyester products) should NOTt be recommended to achieve definitive fascial closure in patients with open abdomen and should be placed only in patients without other alternatives (Grade 1B). - Biologic meshes are reliable for definitive abdominal wall reconstruction in the presence of a large wall defect, bacterial contamination, comorbidities and difficult wound healing. NPWT can be used combined with biologic mesh to facilitate granulation and skin closure (Grade 2B). - Non–cross-linked biologic meshes seem to be preferred in sublay position when the linea alba can be reconstructed. Non–cross-linked biologic mesh is easily integrated, with reduced fibrotic reaction and lesser infection and removal rate (Grade 2B).

Table 2 Statement Grid *(Continued)*

	- The long-term outcome of a bridging non–cross-linked biologic mesh is laxity of the abdominal wall and a high rate of recurrent ventral hernia. In the bridge position (no linea alba closure), cross-linked biologic meshes maybe associated with less ventral hernia recurrence (Grade 2B).
Best treatment for open abdomen and entero-atmospheric fistulas	- Several clinical circumstances may contribute to the development of entero-atmospheric fistula and few risk factors may predict its development. Awareness of this complication and avoidance of contributing conditions for its development are mandatory; moreover preemptive measures are imperative (Grade 1C). - The management of entero-atmospheric fistula should be personalized according to standard classification and grading system. Current different classification schemes echo the problematic and challenging issues related to their management (Grade 1C) - The caloric intake and protein demands of patients with entero-atmospheric fistula increase; the Nitrogen balance should be corrected and protein supplemented. Nutrition should be started immediately upon recognition of entero-atmospheric fistula (Grade 1C) - Entero-atmospheric fistula effluent isolation is essential for proper wound healing. Separating the wound into different compartments in order to facilitate the collection of fistula output is of paramount importance (Grade 2A). - Many methods for wound care exist; however in the presence of entero-atmospheric fistula in open abdomen, negative pressure wound therapy makes effluent isolation feasible and wound healing conceivable (Grade 2A). Definitive management of entero-atmospheric fistula should be delayed to after the patient has recovered and the wound completely healed (Grade 1C).
Nutritional support	- Open abdomen patients are in a hyper-metabolic condition; an immediate and adequate nutritional support is mandatory (Grade 1C). - Open abdomen techniques result in a significant nitrogen loss that must be replaced with a balanced nutrition regimen (Grade 1C). - Early enteral nutrition should be started as soon as possible if the gastrointestinal tract allows (Grade 1C). - Enteral nutrition should be delayed in patients with high output fistula with no possibility to obtain feeding access distal to the fistula (Grade 2C) - Oral feeding is not contraindicated; whenever it's possible it could be started as soon as the patient is able to eat (Grade 2C).
Patient Mobilization	- To date, no recommendations can be made about early mobilization of patients with open abdomen.

cohort studies showed the effectiveness of OA technique in treating severe peritonitis. At present, however, no definitive data from randomized trials exist.

Open abdomen in vascular emergencies

The open abdomen should be strongly considered following management of hemorrhagic vascular catastrophes such as ruptured abdominal aortic aneurysm (grade 1C).

The open abdomen should be considered following surgical management of acute mesenteric ischemic insults (grade 2C).

The ACS has been well described in the setting of ruptured abdominal aortic aneurysm (rAAA) [17]. Rupture of aortic as well as iliac or visceral aneurysm often results in life-threatening hemorrhagic shock. The combination of severe shock and massive resuscitation contributes to retroperitoneal, mesenteric, and bowel wall edema and production of ascites that can increase abdominal pressure and lead to ACS. Intra-abdominal hypertension occurs in up to 50% of patients following AAA repair, and ACS occurs in 8–20%. Mortality after rAAA is as high as 30–50%; of note, mortality is generally twice as high among patients who develop ACS compared with those who do not [18].

Consequently, prevention of ACS, if possible, would be of tremendous benefit to the patient.

In prospective non-randomized studies, the incidence of ACS is reduced when prophylactic OA is employed [19]. Unfortunately, selection criteria for employing OA are not well defined; the surgeon might consider inability to close the fascia without tension; use of aortic balloon occlusion catheter; and preoperative blood loss >5 L [19, 20]. Such criteria should prompt the surgeon to consider temporary OA utilization. When the abdomen is closed primarily, postoperative monitoring of IAP is recommended, with vigilance for ACS as reflected by elevated airway pressures, reduced cardiac output, or oliguria. Concerns for infection of aortic grafts with OA are allayed by existing data, indicating a relatively low rate [21]. Patients are often selected for endovascular repair (EVAR) of rAAA if they have less hemodynamic compromise. Although it is less common, ACS still occurs after EVAR [17]. The major risk factor appears to be massive resuscitation. These patients should have vigilant monitoring for elevated IAP and the onset of ACS.

Mesenteric ischemia may result from arterial (thrombotic, embolic, or low perfusion) or venous (venous thrombosis) insults. Fundamental principles of management include making the diagnosis, restoration of intestinal perfusion, and assessment of bowel viability with resection as necessary. The bowel ischemia leads to bowel wall and mesenteric edema, as well as ascites production; reperfusion of the bowel can exacerbate bowel edema and ascites and thus increase risk of ACS. For this reason, OA use should be considered following restoration of perfusion in a patient with acute mesenteric ischemia. As there are no reliable independent predictors of ACS in this setting, the surgeon should assess bowel swelling and the patient's physiology to make this decision [22, 23]. Another reason to consider temporary OA following mesenteric ischemia is to facilitate second-look laparotomy to assess bowel viability and perform bowel anastomosis as needed [24]. Bowel resection is much less common in the setting of venous thrombosis than arterial occlusion, so the patients with mesenteric venous thrombosis probably do not require OA as often as those with acute arterial occlusion [25]; although, IAP should be followed.

Open abdomen in pancreatitis

In patients with severe acute pancreatitis unresponsive to step-up conservative management, surgical decompression and leaving the abdomen open is effective in treating abdominal compartment syndrome (grade 2C).

Leaving the abdomen open after surgical necrosectomy for infected pancreatic necrosis is not recommended except in those situations at high risk of abdominal compartment syndrome (grade 1C).

Acute pancreatitis (AP) is a mild self-limiting disease in the majority of cases, even though the 15% of patients with AP progress to severe disease identified by development of persistent organ failure [26]. Multiple organ failure (MOF) is the factor mainly associated to mortality in AP, as a counterpart in absence of organ dysfunction or if it transient the risk of dying is very low [27–29]. However, in those with severe AP, MOF develops generally early, with over half of the patients exhibiting organ dysfunction's signs at hospital admission and in any case, most part of them develops within the first 4 days after admission [28, 30]. More than half of the deaths happen within the first week from onset of AP and generally within a week after MOF first symptoms [31]. Principal treatments of MOF are support therapies: vasopressors, fluid replacement, and renal replacement therapy and mechanical ventilation if indicated. During AP, IAH/ACS may aggravate MOF, and therefore, constant IAP measurements are crucial to identify patients with high risk of developing ACS [32]. ACS should be prevented and treated, whenever possible, with non-operative management. Surgical decompression is the last but the most effective tool to decrease the IAP, and it should not be postponed if the patient presents ACS manifestation [5, 33].

In the event of AP, the risk to develop subsequent infections (i.e., bacteremia, pneumonia and infection of pancreatic or peripancreatic necrosis) is increased. The first week of illness is crucial for the extra-pancreatic infection occurrence, whereas pancreatic necrosis usually becomes infected later [34]. Some factors are associated to an increased risk of infected necrosis: the presence of organ failure, early bacteremia, and the extent of pancreatic necrosis [34]. Surgical necrosectomy is the last resort if more conservative management including percutaneous drainage failure [35]. Patients with persistent organ failure complicated with infected pancreatic necrosis face a very high mortality risk [36].

Optimal technique for temporary abdominal closure

Negative pressure wound therapy with continuous fascial traction is suggested as the preferred technique for temporary abdominal closure (grade 1B).

Temporary abdominal closure without negative pressure wound therapy (e.g., mesh alone, Bogota bag) whenever possible should NOT be applied for the purpose of temporary abdominal closure, because of low delayed fascial closure rate and being accompanied by a significant intestinal fistula rate (grade 1B).

There is inadequate evidence to make a recommendation regarding use of negative pressure wound therapy in combination with fluid instillation in patients with temporary abdominal closure (NOT GRADED).

The perceived indications and subsequent treatment choices in managing OA differ among surgeons. The existing techniques result in different risk of entero-atmospheric fistula (EAF) and the different rate of delayed fascial closure. Overall, 74 relevant studies exist for a total of 4358 patients: 3461 (79%) with peritonitis. The described OA indications are considerably different. Thirty-eight out of 78 series described negative pressure wound therapy (NPWT) TAC systems. NPWT with a dynamic component (mesh-mediated fascial traction or dynamic sutures) gives the best results in terms of delayed fascial closure, but dynamic sutures result more often in fistula. NPWT without a dynamic component (Barker's VAC or commercial products) for the use of temporary fascial closure has a moderate delayed fascial closure rate and a fistula rate similar to mesh closure without NPWT.

Several TAC techniques exist that could be used alone or combined together. Six-eight series reported about one TAC technique. Ten series described patients managed with combined TAC systems. NPWT was used alone in 32 studies [37–68], and in 6 studies, NWPT is combined with fascial traction (mesh or sutures) [69–74] and eight series described the use of meshes (non-absorbable and/or absorbable) [75–81]. Six series reported about the Bogota-bag use [75, 82–86]; five, about Zipper [87–91]; and other five, about dynamic retention sutures [92–96]. Two more series described loose packing [97, 98]. Lastly, the Wittmann patch was used in one series [99]. The remnant three series applied different TAC systems [82, 100, 101]. The delayed fascial closure rate ranged from 3.2 to 100%.

Twenty-two series were prospective, and ten out of them described NPWT (608 patients) showing a weighted fascial closure rate of 53.9% and an EAF rate of 9.8%. The four prospective series on NPWT with fascial traction (411 patients) showed a weighted fascial closure rate of 77.8% and an EAF rate of 4.3%. Including retrospective studies data per closure type are in line with the aforementioned results. With the highest weighted fascial closure rate for NPWT with fascial traction (73.1%) and dynamic retention sutures (73.6%). TAC using a mesh or zipper showed the lowest delayed closure rates (34.2 and 34.0% respectively). Nine series were not exhaustive in describing eventual fascial closure attempts [16, 45, 75, 81, 87, 89, 98, 102, 103].

Is there a role for NPWT with fluid instillation?

There are no series published on the use of NPWT with instillation in situations of TAC in non-trauma patients or in trauma patients. Recently, a systematic review performed by an expert consensus group has been published underlining the need of more evidence to support the fluid instillation and giving no recommendation of its use in abdominal wound [104].

Planning re-exploration before definitive closure

In critically ill non-trauma patients with open abdomen, once any requirements for on-going resuscitation have ameliorated, early re-operation with the intention of closing the abdomen should be given a high priority (grade 1C).

In critically ill patients with open abdomen, re-laparotomy with concern for ongoing ischemia/contamination re-operation should be conducted no later than 24–48 h after the index operation, with the duration from the index operation shortening with increasing degrees of patient non-improvement and hemodynamic instability (grade 1C).

A related question for clinicians is when to re-operate (if ever) for the sole purpose of “revise” when there is recognition that closing an abdomen will not be possible. This question may be further conceptually complicated in an attempt to distinguish indications to re-operate because the patient is not improving or deteriorating and there is fear that contamination or ischemia is ongoing and those cases of non-improvement or only modest improvement in whom there is operation intention to “wash” the peritoneal cavity and to “change” the TAC dressing or device. No RCTs or meta-analyses examining the timing of re-operation in OA patients exist. Guidelines and review papers did not generally discuss timing of re-operation [8, 105]. In the position paper of the WSES, it is recommended that as a general principle, patients should be taken back to the operating room at 24–48 h after the initial surgery [2]. Other expert opinions come from the survey of Trauma Association of Canada in 2006, and the majority of responders indicated the best timing included between 24 and 72 h [106, 107]. Pommerening et al. utilized the American Association for the Surgery of Trauma (AAST) Open Abdomen Registry to evaluate time to the first re-operation on trauma OA patients as a predictor of primary fascial closure using a hierarchical multivariate logistic regression analysis [108]. Adjusting for other factors, including resuscitation volumes, increasing delay to the first re-operation was associated with a decreased likelihood of primary fascial closure (PFC), with a 1.1% decrease in PFC rates for every hour after 24 h from the index operation [108]. Further, there was a trend (95% CI 1.0–3.25 OR) of increased complications in patients having the first re-operation after 48 h [108].

It should be clearly understood however that extrapolation of these findings regarding the timing of re-operation in trauma patients might not be directly applicable to non-trauma patients with OA. It is becoming apparent that infected and non-infected patients with auto-activation of the immune responses leading to multi-organ dysfunction syndrome (MODS) and MOF have more fundamental differences than previously appreciated [109]. Fundamental evidences from basic science are emerging justifying the OA in critically ill/injured patents in order to manipulate the systemic immune response and ameliorate the bio mediator burdens of catastrophic illness [110–113]. There are also newly described populations of fully mature indwelling peritoneal macrophages that migrate locally within the peritoneal cavity within an hour of injury [114]. Whether mechanically removing such cell populations through scheduled “wash-outs” is beneficial or harmful is a completely unstudied question. Thus, the timing of re-operation is more complex in non-trauma patients and urgently requires further study. Lastly, in critically ill patients with an OA, re-laparotomy with the intention of cleaning or “washing-out” the abdomen has an unknown priority and should be subjected to future randomized study.

Best timing to definitively close an open abdomen

Fascia should be closed as soon as possible (grade 1C).

Acidosis (pH <7.25), hypothermia (temperature <34 °C), and coagulopathy (TEG, INR) are not predictive of the need for maintaining the open abdomen in non-trauma patients (grade 2A).

The abdomen should be maintained open in non-trauma patients if the source of contamination persists, if a condition of hemodynamic instability persists meaning in the presence of an on-going fluid resuscitation or vasopressor support necessity, if a deferred intestinal anastomosis is needed, if there is the necessity for a planned second look for ischemic intestine, and lastly if there are *concerns about abdominal compartment syndrome development (grade 2C).*

Early fascia closure (within 7 days) should be the strategy for management of the open abdomen once the source control has been reached, the severe sepsis has been controlled meaning that the patient is hemodynamically stable and the hypoperfusion has been definitively corrected, no further surgical re-exploration is needed, and there are no concerns for abdominal compartment syndrome (grade 2C).

The early definitive abdominal closure is the first goal to achieve in order to reduce the OA complications rate [115], (i.e., EAF, fascial retraction with loss of abdominal wall domain, and incisional hernias) [115, 116]. The primary closure rates have a bimodal distribution, with early closure depending on postoperative intensive care management and delayed closure depending on the choice of the TAC technique [117]. Mortality, complications, and length of stay were compared between early and delayed fascial closure in a meta-analysis [118]. 3125 patients were included and 1942 (62%) successfully achieved early fascial closure. Early fascial closure is a factor significantly associated with a reduced mortality (12.3 versus 24.8%, RR 0.53, $P < 0.0001$) and complication rate (RR, 0.68, $P < 0.0001$). Early fascial closure is commonly performed within 4–7 days of the initial laparostomy [13]. No major technical difficulties are described to obtain primary fascial closure within few days from the index operation. Patients having abdominal sepsis are less likely to achieve an early fascial closure [119] and therefore should have closure attempts performed as soon as possible after severe abdominal sepsis is controlled [120].

Best solution to definitively close an open abdomen

Often the OA, particularly if prolonged, results in fascia retraction and consequently in large abdominal wall defects that require complex abdominal wall reconstruction. Moreover, the situation is often complicated by a contaminated field [121] with high risk of infections and wound complications, such as wound infections, seromas, fistula formation, recurrence of the defect, and mortality [122–124].

Non-mesh-mediated techniques

Primary fascia closure is the ideal solution to restore the abdominal closure (grade 2A).

Component separation is an effective technique; however, its early use is NOT recommended in fascial temporary closure. It should be considered only for definitive closure or reconstructive interventions (grade 2C).

*Planned ventral hernia (skin graft or skin closure only) remains an option for complicated open abdomen (*i.e., *in the presence of entero-atmospheric fistula or in cases with a protracted open abdomen due to underlying diseases) or in those low-resource setting where no other facilities are present (grade 2C).*

Abdominal component separation is most commonly considered an elective procedure for ventral hernia repair [118]. One important technique described for the reconstruction of the abdominal wall is the component separation. The technique of anterior component separation consists in a relaxing incision made in the aponeurosis of the external oblique muscle, a separation of the external and internal oblique muscle and the incision of the rectus fascia to achieve the advancement of the abdominal wall to cover the defect. This technique has been well studied and described in elective giant ventral hernia repair, and it provides an effective technique with a recurrence rate of 16% [125, 126] but a very relevant complication rate of 50%. Other surgical techniques that have been described include the posterior component separation: the rectus sheath is opened and the posterior rectus fascia and rectus muscle are separated. At the lateral margin of the rectus muscle, the aponeurosis of the transverse abdominis muscle is incised with the separation of the internal oblique muscle from the transverse abdominis muscle.

However, the use of abdominal component separation technique was recently described in acute fascia closure after open abdomen in a small case series by Rasilainen et al. [127] with 75% of primary fascia closure. At present, there is not enough evidence to support component separation in the acute setting due to the related high morbidity and the fact that these techniques can only be performed on a patient once, so that if ill timed, future options are not available. Therefore, a valuable alternative option for closure of the open abdomen remains the planned ventral hernia: its main goal is to cover abdominal viscera to prevent complications such as EAF. The abdominal wall defect could be closed only with skin suture and or a skin graft put on the underlying granulating tissue creating a planned laxity. After physiologic recovery and a significant period of scar and adhesion maturation, the complete restoration of the patient's abdominal wall through reconstructive techniques can be undertaken as an elective procedure.

Mesh-mediated techniques

A fascial bridge using prosthetic mesh (polypropylene, polytetrafluoruroethylene (PTFE) and polyester products) should not be recommended to achieve definitive fascial closure in patients with open abdomen and should be placed only in patients without other alternatives (grade 1B).

Biologic meshes are reliable for definitive abdominal wall reconstruction in the presence of a large wall defect, bacterial contamination, comorbidities, and difficult wound healing. NPWT can be used combined with *biologic mesh to facilitate granulation and skin closure (grade 2B).*

Non-cross-linked biologic meshes seem to be preferred in sublay position when the linea alba can be reconstructed. Non-cross-linked biologic mesh is easily integrated, with reduced fibrotic reaction and lesser infection and removal rate (grade 2B).

The long-term outcome of a bridging non-cross-linked biologic mesh is laxity of the abdominal wall and a high rate of recurrent ventral hernia. In the bridge position (no linea alba closure), cross-linked biologic meshes maybe associated with less ventral hernia recurrence (grade 2B).

Two meta-analyses exist on BP in abdominal wall defect. The first, by Sharrock et al. investigated the management and closure of OA in trauma patients [128]. Among the included studies, the point estimate recurrence rate of ventral hernia after 1 year of BP positioning was 51%. However, the authors highlighted the small number of included studies and their poor quality; moreover, as above mentioned, great differences exist between trauma and septic patients and great caution should be addressed in interpretation of this result. A systematic review and meta-analysis by Atema et al. [129] investigated the utilization of BP in abdominal wall reconstruction. They clearly stated that the poor quantity and quality of available data strongly limits taking a clear message from the results. Biological material in infected fields had a recurrence rate of 30% compared with 7% of synthetic material, but data were derived from a single study and does not justify the use of synthetic materials, especially as a bridge position after OA.

The "bridging" technique refers to using some mesh (either prosthetic or biologic) to physically interpose between native abdominal wall fascia that either cannot or should not be primarily opposed. Thus, such fascial defects can be closed with a mesh in a bridging position. In general, non-absorbable synthetic materials (i.e., polypropylene mesh) reinforce any fascial repair through a combination of mechanical tension and intense inflammatory reaction, resulting in the entrapment of the mesh into scar tissue. However, in a bridging position, there is no native tissue to protect viscera from the mesh and thus, the persistent inflammatory response combined with the contaminated field may induce local side effects such as adhesions, erosions, and fistula formation [130–135]. International guidelines on emergency repair of abdominal wall hernia therefore do *not* recommend the use of synthetic meshes in contaminated fields [136].

Biological prosthesis (BP) has been designed to perform as permanent surgical prosthesis in the abdominal wall repair, minimizing mesh-related complications [137]. The rationale of their usage in OA is based on the premise that the implantation of a biologic material triggers a cascade of events leading to new healthy tissue deposition and prosthesis remodeling. The presence of vital tissue therefore allows for perfusion and a native immune response preventing mesh infection and abscess formations. The ideal BP will also maintain mechanical characteristics of a synthetic mesh with a sufficient mechanical strength to withstand the physiological and anatomic stresses of the human abdominal wall. Such an ideal BP should also tolerate adjunctive NPWT to facilitate wound healing, granulation, and skin closure [100, 138].

Discordant data have been published about the use of BP to bridge a wide defect of the abdominal wall. The evidence is limited with few studies, all non-randomized, and with an overall small number of cases. Further among heterogeneous patients reported, recurrence rates have ranged between 0 and 100% [139–152]. When used as a bridge to close the fascia defect, the reported recurrence rate in a large retrospective series was >80% [153]. Another study by Booth and colleagues compared primary fascia closure with mesh reinforcement with the use of the mesh as a bridge and demonstrated a higher recurrence rate in the mesh in a bridge position (8 vs. 56%, $p < 0.001$) [154].

Several studies investigated the best anatomical position in terms of BP function, but were not specifically focused on OA reconstruction. Nonetheless, evidence, including that from randomized trials, suggest that implanting the BP in the sublay position results in a lower recurrence and complication rate [155–157]. However, it should be stressed that the data included was not specific for the OA situation and the heterogeneity among patients and indications was very high, resulting in a poor level of evidence.

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A systematic review and meta-analysis by Atema et al. [129] investigated the utilization of BP in abdominal wall reconstruction; the poor quantity and quality of available data strongly limits the results. Biological material in infected fields had a recurrence rate of 30% compared with 7% of synthetic material, but data were derived from a single study and does not justify the use of synthetic materials, especially as a bridge position after OA.

In conclusion, no definitive evidence-based conclusions could be obtained currently from the literature. The

BJORK CLSSIFICATION 2009	
GRADE	**DESCRIPTION**
1 A	Clean OA without adherence between bowel and abdominal wall or fixity
B	Contaminated OA without adherence/fixity
2 A	Clean OA developing adherence/fixity
B	Contaminated OA developing adherence/fixity
3 A	OA complicated by fistula formation
4	Frozen OA with adherent/fixed bowel; unable to close surgically; with or without fistula

BJORK CLASSIFICATION 2016	
GRADE	**DESCRIPTION**
1 A	Clean OA without adherence between bowel and abdominal wall or fixity
B	Contaminated OA without adherence/fixity
C	Enteric leak, no fixation
2 A	Clean OA developing adherence/fixity
B	Contaminated OA developing adherence/fixity
C	Enteric leak, developing fixation
3 A	Clean, frozen abdomen
B	Contaminated, frozen abdomen
4	Established enteroatmospheric fistula, frozen abdomen

Fig. 1 Open Abdomen classification according to Bjork et al. [168]

available evidence is really weak: most of the cited meta-analysis included rather poor quality retrospective case series. There is also great heterogeneity among the indications for mesh implantation, the anatomic positioning of the mesh, and the type of mesh. This further weakens the quality of the evidences. Thus, well-performed randomized trials comparing different type of meshes and the techniques of mesh positioning are urgently required.

Best treatment for open abdomen and entero-atmospheric fistulas

Several clinical circumstances may contribute to the development of entero-atmospheric fistula and few risk factors may predict its development. Awareness of this complication and avoidance of contributing conditions for its development are mandatory; moreover, preemptive measures are imperative (grade 1C).

The management of entero-atmospheric fistula should be personalized according to standard classification and grading system. Current different classification schemes echo the problematic and challenging issues related to their management (grade 1C).

The caloric intake and protein demands of patients with entero-atmospheric fistula increase; the nitrogen balance should be corrected and protein supplemented. Nutrition should be started immediately upon recognition of entero-atmospheric fistula (grade 1C).

Entero-atmospheric fistula effluent isolation is essential for proper wound healing. Separating the wound into different compartments in order to facilitate the collection of fistula output is of paramount importance (grade 2A).

Many methods for wound care exist; however, in the presence of entero-atmospheric fistula in an open abdomen, negative pressure wound therapy makes effluent isolation feasible and wound healing conceivable (grade 2A).

Definitive management of entero-atmospheric fistula should be delayed to after the patient has recovered and the wound completely healed (grade 1C).

Enteric fistula is a severe complication following abdominal surgery. The opening of a fistula onto dehisced wound therefore exposing and communicating the bowel and its effluent to the atmosphere is defined as EAF. The incidence of EAF varies from 4.5 to 25% in the trauma setting [158] and from 5.7 and 17.2% in non-trauma patients [105]. The presence of this complication dramatically increases considerably mortality, length of stays, and costs [159].

Many factors may contribute to the development of EAF. All linked as a "vicious cycle": the lack of overlying soft tissue, with its blood supply, precludes spontaneous healing and the exposed viscera predispose to additional disruptions in the gastrointestinal tract. EAFs may result from various etiologies: anastomotic dehiscence or disruption, iatrogenic injury during dissection or inappropriate handling, and presence of synthetic prosthetic material (i.e., mesh) and from the prolonged exposure of bowel [160–163]. ACS and severe IAH may result in reduced bowel blood supply and therefore contribute to EAF development [68]. A prospective analysis of 517 trauma emergency laparotomies showed that large bowel resections, large volume fluid resuscitation (>5 L/24 h), and increased number of re-explorations were significantly associated with an increased incidence of EAF

[158]. Preemptive measures could be undertaken in order to prevent this complication: early abdominal wall closure, bowel coverage with omentum or skin, and no direct application of NPWT on the viscera are some of these measures [112, 164, 165].

Several classifications and grading systems of EAF exist. Schein and Decker proposed in 1991 a grading system based on the fistula location. Grade IV indicates a fistula related to large abdominal wall defects with grades IVa and IVb indicating the site of the fistula in regards to its location [166]. EAF can be classified based on the fistula effluent output: low (<200 ml/day), moderate (200-500 ml/day), and high (>500 ml/day) [167]. Bjork et al. proposed a classification based on the presence of adhesions of the bowel in the setup of the open abdomen as well as the association to the fistula formation (Fig. 1), and this was later adapted by WSACS [168]. Di Saverio et al. proposed a comprehensive classification based on the combination of different criteria as anatomical location, output, exposure, and number of fistulas [169]. As a general principle, a single, superficial fistula located in the lower GI tract with a low output has a higher probability of spontaneous closure rather than multiple fistulas deep in the wound with high output [169, 170]. According to this principle, the management should be tailored to each clinical situation and individualized accordingly. In conclusion, the presence of several different classifications represents the true difficulties in the management of EAF in OA. Level of evidence is poor and many recommendations are based on expert opinion suggestions.

EAF is a poorly predictable and, above all, avoidable complication. When patients develop EAF, an accurate and tailored management scheme should be adopted. Nutrition plays a key role in the management of these patients and should be always kept in mind as a fundamental part of the treatment. The open abdomen strategy may result in fluid and electrolytes loss resulting in acid-base derangements [8]. The anatomy and the characteristics of the EAF(s) should be defined in order to plan the best treatment option [171]. Parenteral nutrition (TPN) should be started immediately after the patient resuscitation. Enteral nutrition in OA patients has been well studied demonstrating a reduction in infectious complications preserving the intestinal mucosal barrier and its immunological function [172–174]. Enteral nutrition in patients with an EAF is has but may increase fistula output. Only small series of patients with EAF treated with EN exists; therefore, no strong evidence can support these treatments and further studies are needed [175, 176]. The use of octreotide analogs is controversial. No evidence exists about the use of somatostatin and octreotide in managing of EAF. Few studies suggest that octreotide may reduce fistula output by diminishing GI secretions [177] while others argue their benefit due to this agents' reduction in splanchnic blood flow and reduction in immune function [178, 179].

The main goal in the management of EAF should be the closure of the fistula. Differently from common GI fistulas, the EAF is not a true fistula since a fistula tract does not exist. The lack of surrounding tissues prevents the spontaneous closure. The goal of the treatment should be focused on trying to isolate the fistula effluent and enhancing the formation of granulation tissues surrounding it. Several different techniques were described and proposed in the literature to control and treat EAF, and some attempts to standardize its management exist [169, 170]. A patient diagnosed with EAF in the setup of OA should be treated by medical personnel familiar with this complication and its consequences.

Accurate fistula definition and anatomy should be made. Sepsis control and management is important. Diversion of the fistula output in order to maintain clean the peritoneal cavity is mandatory. Fistula effluent should be measured in order to facilitate fluid balance and to ensure skin protection from its digestive nature on the skin. This will enhance and allow better patient care and mobility.

Several different dressing and techniques were described for the management of EAF, each one with relatively small case series and discordant results with a consequent poor level of evidence [162, 170, 180–183]. Proposed treatments vary from primary suture and fibrin glue for small exposed distal fistula to a fistula suspension fixating the fistula edges to the skin. Several variants of NPWT with devices for fistula isolation and diversion were described with promising outcomes.

The several techniques are described in detail elsewhere and are not in the scope of the current position paper [170]. The described method to manage NPWT in patients with EAF in the setup of OA should be applied depending on surgeon preference, skills, and expertise and according to hospital facilities and material availability. Generally, negative pressure wound therapy, with specifically described variants, is the most accepted technique. EAF isolation and proper wound management will enable skin grafting and converting EAF to a more controllable one with ease of applying effluent collection bag. The definitive treatment, i.e., closure of the fistula and repairing the abdominal wall defect should be postponed at least 6 months and only after the patient and the wound healed completely.

Nutritional support

Open abdomen patients are in a hyper-metabolic condition; an immediate and adequate nutritional support is mandatory (grade 1C).

Open abdomen techniques result in a significant nitrogen loss that must be replaced with a balanced nutrition regimen (grade 1C).

Early enteral nutrition should be started as soon as possible if the gastrointestinal tract allows (grade 1C).

Enteral nutrition should be delayed in patients with high output fistula with no possibility to obtain feeding access distal to the fistula (grade 2C).

Oral feeding is not contraindicated; whenever its possible, it could be started as soon as the patient is able to eat (grade 2C).

The hyper-catabolic state of critically ill patients is associated with muscle proteolysis, acute protein malnutrition, immune function impairment, and subclinical development of MOF. Several studies clearly demonstrated malnutrition as a fundamental risk factor associated to poor outcomes during hospital stay [184]. Furthermore, in a critically ill patient, OA leads to significant nitrogen loss estimated to be 2 g per liter of abdominal fluid output. This issue requires adequate consideration and an adjusted integration [185]. For this reason, the measurement of the abdominal fluid loss is mandatory [185]. This loss in nitrogen and protein is ulterior greatly increased in the presence of EAF. A particular attention must be given to this critical aspect because patients with OA are the sickest, most inflamed, and subsequently most hypermetabolic among surgical patients. During the OA patient management, once the resuscitation is almost completed and the GI tract allows it, EN should be started as soon as possible. Thus, it will bring beneficial effects for the patient as faster fascia closure and lower pneumonia and fistula rate [173, 186, 187]. If malnutrition occurs, mucosal atrophy and malabsorption are among the earliest consequences. Gut-associated lymphoid tissue seems to be diminished, and as a consequence, it can increase the risk for disseminated infection due to bacterial translocation through the intestinal wall [188]. EN helps in maintaining gut mucosal barrier in good shape and function; as a consequence, it has been demonstrated to enhance immunity and IgA secretion, to prevent muscle atrophy, and lastly to decreases systemic inflammation and oxidative injury [188, 189]. Early EN within the first 24–48 h is demonstrated to improve wound healing, decrease catabolism, preserve GI tract integrity, and finally, it reduces complications, length of hospital stay, and costs. Compared to TPN early EN decreases septic complications especially in abdominal trauma and traumatic brain injuries. A retrospective, single-institution study comparing DCS interventions with open abdomen performed to treat ACS, 43 patients underwent early (<4 days) and 35 late (>4 days) EN. Early EN significantly increased primary closure (74% vs. 49%), reduced the fistula rate (9% vs. 26%) with no difference in infections and but with a significant reductions in hospitalization costs [186].

Patient mobilization

To date, no recommendations can be made about early mobilization of patients with open abdomen.

Patients with an open abdomen generally should not be mobilized out of bed until their abdomens are definitively closed, for risk of evisceration [190]. This statement was extrapolated from trauma literature [191]. However, prolonged bed rest is associated with significant increase in complication rate. More recent attention has been focused on intensive care unit (ICU)-acquired weakness and the long-term adverse functional sequelae for ICU survivors, particularly in the physical domain and this has led to an increased interest in early mobilization in the ICU as a potential means of prevention [192–196]. The optimal timing for initiation of mobilization of patients with OA has yet to be defined. Early mobilization is currently defined as occurring within the first 2 to 5 days of ICU admission [197].

Patients with open abdomen managed with NPWT however, may be mobilized by active or passive transfer. Further research must occur to provide the rationale to early mobilization prior to definitive abdominal closure.

Conclusions

Management of the open abdomen remains a very controversial domain, in which many techniques are still debated. Many important issues remain to be addressed through carefully designed and rigorously conducted studies. Until better data is available, the use of the OA should be carefully tailored to each single patient taking care to not overuse this effective tool. Every effort should be exerted to attempt abdominal closure as soon as the patient can physiologically tolerate it. Finally, all the precautions should be considered to minimize the complication rate.

Abbreviations

AAST: American Association for the Surgery of Trauma; ACS: Abdominal compartment syndrome; AP: Acute pancreatitis; BP: Biological prosthesis; EAF: Entero-atmospheric fistula; EN: Enteral nutrition; EVAR: Endovascular repair; GRADE: Grading of Recommendations Assessment, Development and Evaluation; IAH: Intra-abdominal hypertension; IAP: Intra-abdominal pressure; INR: International Normalized Ratio; MODS: Multi-organ dysfunction syndrome; MOF: Multiple organ failure; NPWT: Negative pressure wound therapy; OA: Open abdomen procedure; PTFE: Polytetrafluoruroethylene; rAAA: Ruptured abdominal aortic aneurysm; RCT: Randomized controlled trial; TAC: Temporal abdominal closure; TEG: Thromboelastography; TPN: Parenteral nutrition; WSACS: The Abdominal Compartment Syndrome; WSES: World Society of Emergency Surgery

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Competing interest
Manu LNG Malbrain is Founding President and current Treasurer of the Abdominal Compartment Society (WSACS, www.wsacs.org). He is also a member of the executive committee of the International Fluid Academy (IFA). The IFA is integrated within the not-for-profit charitable organization iMERiT (International Medical Education and Research Initiative) under Belgian Law. The IFA website (http://www.fluidacademy.org) is now an official SMACC (Social Media and Critical Care) affiliated site, and its content is based on the philosophy of FOAM (Free Open Access Medical Education—#-FOAMed). He is a member of the medical advisory board of Pulsion Medical Systems (Maquet Getinge group) and consults for Acelity, ConvaTec, Spiegelberg and Holtech Medical.
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References
1. Bailey J, Shapiro MJ. Abdominal compartment syndrome. Crit Care. 2000;4:23–9.
2. Sartelli M, Abu-Zidan FM, Ansaloni L, Bala M, Beltrán MA, Biffl WL, et al. The role of the open abdomen procedure in managing severe abdominal sepsis: WSES position paper. World J Emerg Surg. 2015;10:35.
3. Van Hee R. Historical highlights in concept and treatment of abdominal compartment syndrome. Acta Clin Belg. 2007;62:9–15.
4. Demetriades D. Total management of the open abdomen. Int Wound J. 2012;9:17–24.
5. Kirkpatrick AW, Roberts DJ, De Waele J, Jaeschke R, Malbrain MLNG, De Keulenaer B, et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med. 2013;39:1190–206.
6. Leppäniemi AK. Laparostomy: why and when? Crit Care. 2010;14:216.
7. Ivatury RR. Update on open abdomen management: achievements and challenges. World J Surg. 2009;33:1150–3.
8. Coccolini F, Biffl W, Catena F, Ceresoli M, Chiara O, Cimbanassi S, et al. The open abdomen, indications, management and definitive closure. World J Emerg Surg. 2015;10:32.
9. Sartelli M, Catena F, Ansaloni L, Coccolini F, Corbella D, Moore EE, et al. Complicated intra-abdominal infections worldwide: the definitive data of the CIAOW Study. World J Emerg Surg. 2014;9:37.
10. van Ruler O, Mahler CW, Boer KR, Reuland EA, Gooszen HG, Opmeer BC, et al. Comparison of on-demand vs planned relaparotomy strategy in patients with severe peritonitis: a randomized trial. JAMA. 2007;298:865–72.
11. Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an american college of chest physicians task force. Chest. 2006;129:174–81.
12. Moore LJ, Moore FA. Epidemiology of sepsis in surgical patients. Surg Clin North Am. 2012;92:1425–43.
13. Ordóñez CA, Sánchez ÁI, Pineda JA, Badiel M, Mesa R, Cardona U, et al. Deferred primary anastomosis versus diversion in patients with severe secondary peritonitis managed with staged laparotomies. World J Surg. 2010;34:169–76.
14. Plantefeve G, Hellmann R, Pajot O, Thirion M, Bleichner G, Mentec H. Abdominal compartment syndrome and intraabdominal sepsis: two of the same kind? Acta Clin Belg. 2007;62:162–7.
15. Schein M. Surgical management of intra-abdominal infection: is there any evidence? Langenbeck's Arch Surg. 2002;387:1–7.
16. Robledo FA, Luque-de-León E, Suárez R, Sánchez P, De-la-Fuente M, Vargas A, et al. Open versus closed management of the abdomen in the surgical treatment of severe secondary peritonitis: a randomized clinical trial. Surg Infect. 2007;8:63–72.
17. Rubenstein C, Bietz G, Davenport DL, Winkler M, Endean ED. Abdominal compartment syndrome associated with endovascular and open repair of ruptured abdominal aortic aneurysms. J Vasc Surg. 2015;61:648–54.
18. Reite A, Soreide K, Ellingsen CL, Kvaløy JT, Vetrhus M. Epidemiology of ruptured abdominal aortic aneurysms in a well-defined Norwegian population with trends in incidence, intervention rate, and mortality. J Vasc Surg. 2015;61:1168–74.
19. Ersryd S, Djavani-Gidlund K, Wanhainen A, Björck M. Abdominal compartment syndrome after surgery for abdominal aortic aneurysm: a Nationwide Population Based Study. Eur J Vasc Endovasc Surg. 2016;52:158–65.
20. Björck M. Management of the tense abdomen or difficult abdominal closure after operation for ruptured abdominal aortic aneurysms. Semin Vasc Surg. 2012;25:35–8.
21. Acosta S, Wanhainen A, Bjorck M. Temporary abdominal closure after abdominal aortic aneurysm repair: a systematic review of contemporary observational studies. Eur J Vasc Endovasc Surg. 2016;51:371–8.

22. Kougias P, Lau D, El Sayed HF, Zhou W, Huynh TT, Lin PH. Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. J Vasc Surg Off Publ Soc Vasc Surg [and] Int Soc Cardiovasc Surgery, North Am Chapter 2007;46:467–474.
23. Tilsed JVT, Casamassima A, Kurihara H, Mariani D, Martinez I, Pereira J, et al. ESTES guidelines: acute mesenteric ischaemia. Eur J Trauma Emerg Surg. 2016;42:253–70.
24. Bruns BR, Ahmad SA, O'Meara L, Tesoriero R, Lauerman M, Klyushnenkova E, et al. Nontrauma open abdomens: a prospective observational study. J Trauma Acute Care Surg. 2016;80:631–6.
25. Schermerhorn ML, Giles KA, Hamdan AD, Wyers MC, Pomposelli FB. Mesenteric revascularization: management and outcomes in the United States, 1988–2006. J Vasc Surg. NIH Public Access; 2009;50:341–348.e1.
26. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102–11.
27. Halonen KI, Pettilä V, Leppäniemi AK, Kemppainen EA, Puolakkainen PA, Haapiainen RK. Multiple organ dysfunction associated with severe acute pancreatitis. Crit Care Med. 2002;30:1274–9.
28. Buter A, Imrie CW, Carter CR, Evans S, McKay CJ. Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg. 2002;89:298–302.
29. Mofidi R, Duff MD, Wigmore SJ, Madhavan KK, Garden OJ, Parks RW. Association between early systemic inflammatory response, severity of multiorgan dysfunction and death in acute pancreatitis. Br J Surg. 2006;93:738–44.
30. Mentula P, Kylänpää-Bäck M-L, Kemppainen E, Takala A, Jansson S, Kautiainen H, et al. Decreased HLA (human leucocyte antigen)-DR expression on peripheral blood monocytes predicts the development of organ failure in patients with acute pancreatitis. Clin Sci (Lond). 2003;105:409–17.
31. Mole DJ, Olabi B, Robinson V, Garden OJ, Parks RW. Incidence of individual organ dysfunction in fatal acute pancreatitis: analysis of 1024 death records. HPB. 2009;11:166–70.
32. De Waele JJ, Leppäniemi AK. Intra-abdominal hypertension in acute pancreatitis. World J Surg. 2009;33:1128–33.
33. Mentula P, Hienonen P, Kemppainen E, Puolakkainen P, Leppäniemi A. Surgical decompression for abdominal compartment syndrome in severe acute pancreatitis. Arch Surg. 2010;145:764–9.
34. Besselink MG, Van Santvoort HC, Boermeester MA, Nieuweohuijs VB, Van Goor H, Dejong CHC, et al. Timing and impact of infections in acute pancreatitis. Br J Surg. 2009;96:267–73.
35. van Santvoort HC, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med. 2010;362:1491–502.
36. Petrov MS, Shanbhag S, Chakraborty M, Phillips ARJ, Windsor JA. Organ failure and infection of pancreatic necrosis as determinants of mortality in patients with acute pancreatitis. Gastroenterology. 2010;139:813–20.
37. Tsuei BJ, Skinner JC, Bernard AC, Kearney PA, Boulanger BR. The open peritoneal cavity: etiology correlates with the likelihood of fascial closure. Am Surg. 2004;70:652–6.
38. Rao M, Burke D, Finan PJ, Sagar PM. The use of vacuum-assisted closure of abdominal wounds: a word of caution. Color Dis. 2007;9:266–8.
39. Carlson GL, Patrick H, Amin AI, McPherson G, MacLennan G, Afolabi E, et al. Management of the open abdomen: a national study of clinical outcome and safety of negative pressure wound therapy. Ann Surg. 2013;257:1154–9.
40. Pliakos I, Papavramidis TS, Michalopoulos N, Deligiannidis N, Kesisoglou I, Sapalidis K, et al. The value of vacuum-assisted closure in septic patients treated with laparostomy. Am Surg. 2012;78:957–61.
41. Barker DE, Green JM, Maxwell RA, Smith PW, Mejia VA, Dart BW, et al. Experience with vacuum-pack temporary abdominal wound closure in 258 trauma and general and vascular surgical patients. J Am Coll Surg. 2007;204:784–92.
42. Oetting P, Rau B, Schlag PM. Abdominal vacuum device with open abdomen. Chirurg. 2006;77(586):588–93.
43. Kritayakirana K, Maggio PM, Brundage S, Purtill MA, Staudenmayer K, Spain DA. Outcomes and complications of open abdomen technique for managing non-trauma patients. J Emerg Trauma Shock. 2010;3:118–22.
44. Prichayudh S, Sriussadaporn S, Samorn P, Pak-Art R, Sriussadaporn S, Kritayakirana K, et al. Management of open abdomen with an absorbable mesh closure. Surg Today. 2011;41:72–8.
45. Adkins AL, Robbins J, Villalba M, Bendick P, Shanley CJ. Open abdomen management of intra-abdominal sepsis. Am Surg. 2004;70:137–40.
46. Amin AI, Shaikh IA. Topical negative pressure in managing severe peritonitis: a positive contribution? World J Gastroenterol. 2009;15:3394–7.
47. Bosscha K, Hulstaert PF, Visser MR, van Vroonhoven TJ, van der Werken C. Open management of the abdomen and planned reoperations in severe bacterial peritonitis. Eur J Surg. 2000;166:44–9.
48. Cipolla J, Stawicki SP, Hoff WS, McQuay N, Hoey BA, Wainwright G, et al. A proposed algorithm for managing the open abdomen. Am Surg. 2005;71:202–7.
49. López-Quintero L, Evaristo-Méndez G, Fuentes-Flores F, Ventura-González F, Sepúlveda-Castro R. Treatment of open abdomen in patients with abdominal sepsis using the vacuum pack system. Cir Cir. 2010;78:317–21.
50. Padalino P, Dionigi G, Minoja G, Carcano G, Rovera F, Boni LDR. Fascia-to-fascia closure with abdominal topical negative pressure for severe abdominal infections: preliminary results in a department of general surgery and intensive care unit. Surg Infect. 2010;11:523–52.
51. Schmelzle M, Alldinger I, Matthaei H, Aydin F, Wallert I, Eisenberger CF, et al. Long-term vacuum-assisted closure in open abdomen due to secondary peritonitis: a retrospective evaluation of a selected group of patients. Dig Surg. 2010:329–35.
52. Pliakos I, Michalopoulos N, Papavramidis TS, Arampatzi S, Diza-Mataftsi E, Papavramidis S. The effect of vacuum-assisted closure in bacterial clearance of the infected abdomen. Surg Infect. 2014;15:18–23.
53. Wondberg D, Larusson HJ, Metzger U, Platz A, Zingg U. Treatment of the open abdomen with the commercially available vacuum-assisted closure system in patients with abdominal sepsis: low primary closure rate. World J Surg. 2008;32:2724–9.
54. Khan A, Hsee L, Mathur S, Civil I. Damage-control laparotomy in nontrauma patients: review of indications and outcomes. J Trauma Acute Care Surg. 2013;75:365–8.
55. Brock WB, Barker DE, Burns RP. Temporary closure of open abdominal wounds: the vacuum pack. Am Surg. 1995;61:30–5.
56. Caro A, Olona C, Jimenez A, Vadillo J, Feliu F, Vicente V. Treatment of the open abdomen with topical negative pressure therapy: a retrospective study of 46 cases. Int Wound J. 2011;8:274–9.
57. Fieger AJ, Schwatlo F, Mündel DF-X, Schenk M, Hemminger F, Kirchdorfer B, et al. Abdominal vacuum therapy for the open abdomen—a retrospective analysis of 82 consecutive patients. Zentralbl Chir. 2011;136:56–60.
58. Horwood J, Akbar F, Maw A. Initial experience of laparostomy with immediate vacuum therapy in patients with severe peritonitis. Ann R Coll Surg Engl. 2009;91:681–7.
59. Ozguc H, Paksoy E, Ozturk E. Temporary abdominal closure with the vacuum pack technique: a 5-year experience. Acta Chir Belg. 2008;108:414–9.
60. Perez D, Wildi S, Demartines N, Bramkamp M, Koehler C, Clavien PA. Prospective evaluation of vacuum-assisted closure in abdominal compartment syndrome and severe abdominal sepsis. J Am Coll Surg. 2007;205:586–92.
61. Pérez Domínguez L, Pardellas Rivera H, Cáceres Alvarado N, López Saco Á, Rivo Vázquez Á, Casal NE. Vacuum assisted closure: utilidad en el abdomen abierto y cierre diferido. Experiencia en 23 pacientes. Cir Esp. 2012;90:506–12.
62. Plaudis H, Rudzats A, Melberga L, Kazaka I, Suba O, Pupelis G. Abdominal negative-pressure therapy: a new method in countering abdominal compartment and peritonitis—prospective study and critical review of literature. Ann Intensive Care. 2012;2(Suppl 1):S23.
63. Shaikh IA, Ballard-Wilson A, Yalamarthi S, Amin AI. Use of topical negative pressure in assisted abdominal closure does not lead to high incidence of enteric fistulae. Color Dis. 2010;12:931–4.
64. Smith LA, Barker DE, Chase CW, Somberg LB, Bradford Brock W, Phillip BR. Vacuum pack technique of temporary abdominal closure: a four-year experience. Am Surg. 1997;63:1102–8.
65. Wilde JW, Loudon MA. Modified opsite® sandwich for temporary abdominal closure: a non-traumatic experience. Ann R Coll Surg Engl. 2007;89:57–61.
66. Zielinski MD, Goussous N, Schiller HJ, Jenkins D. Chemical components separation with botulinum toxin A: a novel technique to improve primary fascial closure rates of the open abdomen. Hernia. 2013;17:101–7.
67. Bertelsen CA, Fabricius R, Kleif J, Kristensen B, Gögenur I. Outcome of negative-pressure wound therapy for open abdomen treatment after nontraumatic lower gastrointestinal surgery: analysis of factors affecting delayed fascial closure in 101 patients. World J Surg. 2014;38:774–81.
68. Richter S, Dold S, Doberauer JP, Mai P, Schuld J. Negative pressure wound therapy for the treatment of the open abdomen and incidence

of enteral fistulas: a retrospective bicentre analysis. Gastroenterol Res Pract. 2013;2013:6–11.
69. Fortelny RH, Hofmann A, Gruber-Blum S, Petter-Puchner AH, Glaser KS. Delayed closure of open abdomen in septic patients is facilitated by combined negative pressure wound therapy and dynamic fascial suture. Surg Endosc Other Interv Tech. 2014;28:735–40.
70. Kafka-Ritsch R, Birkfellner F, Perathoner A, Raab H, Nehoda H, Pratschke J, et al. Damage control surgery with abdominal vacuum and delayed bowel reconstruction in patients with perforated diverticulitis Hinchey III/IV. J Gastrointest Surg. 2012;16:1915–22.
71. Kleif J, Fabricius R, Bertelsen CA, Bruun J, Gögenur I. Promising results after vacuum-assisted wound closure and mesh-mediated fascial traction. Dan Med J. 2012;59
72. Kafka-Ritsch R, Zitt M, Schorn N, Stroemmer S, Schneeberger S, Pratschke J, et al. Open abdomen treatment with dynamic sutures and topical negative pressure resulting in a high primary fascia closure rate. World J Surg. 2012;36:1765–71.
73. Acosta S, Bjarnason T, Petersson U, Pålsson B, Wanhainen A, Svensson M, et al. Multicentre prospective study of fascial closure rate after open abdomen with vacuum and mesh-mediated fascial traction. Br J Surg. 2011;98:735–43.
74. Huang Q, Zhao R, Yue C, Wang W, Zhao Y, Ren J, et al. Fluid volume overload negatively influences delayed primary facial closure in open abdomen management. J Surg Res. 2014;187:122–7.
75. García Iñiguez JA, Orozco CF, Muciño Hernández MI, Ortega AL, Trabaldo SS, Cortés Flores AO, Hermosillo Sandoval JMOA. Complications of the management of secondary peritonitis with contained-open abdomen. Comparison of the Bogota's bag vs polypropylene mesh. Rev Gastroenterol Mex. 2004;69:147–55.
76. Schein M. Planned reoperations and open management in critical intra-abdominal infections: prospective experience in 52 cases. World J Surg. 1991;15:537–45.
77. Gentile AT, Feliciano PD, Mullins RJ, Crass RA, Eidemiller LR, Sheppard BC. The utility of polyglycolic acid mesh for abdominal access in patients with necrotizing pancreatitis. J Am Coll Surg. 1998;186:313–8.
78. Losanoff JE, Kjossev KT. Mesh-foil laparostomy. J Am Coll Surg. 1997;185:89–92.
79. Töns C, Schachtrupp A, Rau M, Mumme T, Schumpelick V. Abdominal compartment syndrome: prevention and treatment. Chirurg. 2000;71:918–26.
80. Schachtrupp A, Höer J, Töns C, Klinge U, Reckord U, Schumpelick V. Intra-abdominal pressure: a reliable criterion for laparostomy closure? Hernia. 2002;6:102–7.
81. Sökmen S, Atila K, Bora S, Astarcloğlu H, Çoker A, Füzün M. Evaluation of prosthetic mesh closure in semiopen-abdomen patients. Hernia. 2002;6:124–9.
82. Martinez-Ordaz JL, Cruz-Olivo PA, Chacon-Moya E, de la Fuente-Lira M, Chavelas-Lluck M, Blanco-Benavides R. Management of the abdominal wall in sepsis. Comparison of two techniques. Rev Gastroenterol Mex. 2004;69:88–93.
83. Gönüllü D, Köksoy FN, Demiray O, Özkan SG, Yücel T, Yücel O. Laparostomy in patients with severe secondary peritonitis. Ulus Travma ve Acil Cerrahi Derg. 2009;15:52–7.
84. Kirshtein B, Roy-Shapira A, Lantsberg L, Mizrahi S. Use of the "Bogota bag" for temporary abdominal closure in patients with secondary peritonitis. Am Surg. 2007;73:249–52.
85. Manterola C, Moraga J, Urrutia S. Contained laparostomy with a Bogota bag. Results of case series. Cir Esp. 2011;89:379–85.
86. Doyon A, Devroede G, Viens D, Saito S, Rioux A, Echavé V, et al. A simple, inexpensive, life-saving way to perform iterative laparotomy in patients with severe intra-abdominal sepsis. Color Dis. 2001;3:115–21.
87. Hakkiluoto A, Hannukainen J. Open management with mesh and zipper of patients with intra-abdominal abscesses or diffuse peritonitis. Eur J Surg. 1992;158:403–5.
88. Hubens G, Lafaire C, De Praeter M, Ysebaert D, Vaneerdeweg W, Heytens L, et al. Staged peritoneal lavages with the aid of a Zipper system in the treatment of diffuse peritonitis. Acta Chir Belg. 1994;94:176–9.
89. Hedderich GS, Wexler MJ, McLean AP, Meakins JL. The septic abdomen: open management with Marlex mesh with a zipper. Surgery. 1986;99:399–408.
90. Zingales F, Moschino P, Carniato S, Fabris G, Vittadello F, Corsini A. Laparostomy in the treatment of severe peritonitis: a review of 60 cases. Chir Ital. 2001;53:821–6.
91. Ercan F, Korkmaz A, Aras N. The zipper-mesh method for treating delayed generalized peritonitis. Surg Today. 1993;23:205–14.
92. Koniaris LG, Hendrickson RJ, Drugas G, Abt P, Schoeniger LO. Dynamic retention: a technique for closure of the complex abdomen in critically ill patients. Arch Surg. 2001;136:1359–62. discussion 1363
93. Reimer MW, Yelle JD, Reitsma B, Doumit G, Allen MA, Bell MS. Management of open abdominal wounds with a dynamic fascial closure system. Can J Surg. 2008;51:209–14.
94. Salman AE, Yetisir F, Aksoy M, Tokac M, Yildirim MB, Kilic M. Use of dynamic wound closure system in conjunction with vacuum-assisted closure therapy in delayed closure of open abdomen. Hernia. 2014;18:99–104.
95. Verdam FJ, Dolmans DEJGJ, Loos MJ, Raber MH, De Wit RJ, Charbon JA, et al. Delayed primary closure of the septic open abdomen with a dynamic closure system. World J Surg. 2011;35:2348–55.
96. Haddock C, Konkin DE, Blair NP. Management of the open abdomen with the abdominal reapproximation anchor dynamic fascial closure system. Am J Surg. 2013;205:528–33.
97. Anderson ED, Mandelbaum DM, Ellison EC, Carey LC, Cooperman M. Open packing of the peritoneal cavity in generalized bacterial peritonitis. Am J Surg. 1983;145:131–5.
98. Hollender LF, Bur F, Schwenck D, Pigache P. The "left-open abdomen". Technic, indication and results. Chirurg. 1983;54:316–9.
99. Wittmann DH. Staged abdominal repair: development and current practice of an advanced operative technique for diffuse suppurative peritonitis. Acta Chir Austriaca. 2000;32:171–8.
100. Dietz UA, Wichelmann C, Wunder C, Kauczok J, Spor L, Strauß A, et al. Early repair of open abdomen with a tailored two-component mesh and conditioning vacuum packing: a safe alternative to the planned giant ventral hernia. Hernia. 2012;16:451–60.
101. Losanoff J, Kjossev K. Palisade dorsoventral lavage for neglected peritonitis. Am J Surg. 1997;173:134–5.
102. Ivatury RR, Nallathambi M, Rao PM, Rohman M, Stahl WM. Open management of the septic abdomen: therapeutic and prognostic considerations based on APACHE II. Crit Care Med. 1989;17:511–7.
103. Wittmann DH, Aprahamian C, Bergstein JM. Etappenlavage: advanced diffuse peritonitis managed by planned multiple laparotomies utilizing zippers, slide fastener, and Velcro analogue for temporary abdominal closure. World J Surg. 1990;14:218–26.
104. Kim PJ, Attinger CE, Olawoye O, Crist BD, Gabriel A, Galiano RD, et al. Negative pressure wound therapy with instillation: review of evidence and recommendations. Wounds a Compend Clin Res Pract. 2015;27:S2–19.
105. Atema JJ, Gans SL, Boermeester MA. Systematic review and meta-analysis of the open abdomen and temporary abdominal closure techniques in non-trauma patients. World J Surg. 2015;39:912–25.
106. Karmali S, Evans D, Laupland KB, Findlay C, Ball CG, Bergeron E, et al. To close or not to close, that is one of the questions? Perceptions of Trauma Association of Canada surgical members on the management of the open abdomen. J Trauma. 2006;60:287–93.
107. Kirkpatrick AW, Laupland KB, Karmali S, Bergeron E, Stewart TC, Findlay C, et al. Spill your guts! Perceptions of Trauma Association of Canada member surgeons regarding the open abdomen and the abdominal compartment syndrome. J Trauma. 2006;60:279–86.
108. Pommerening MJ, Dubose JJ, Zielinski MD, Phelan HA, Scalea TM, Inaba K, et al. Time to first take-back operation predicts successful primary fascial closure in patients undergoing damage control laparotomy. Surg (United States). 2014;156:431–8.
109. Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, et al. The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016;315:801–10.
110. Emr B, Sadowsky D, Azhar N, Gatto LA, An G, Nieman GF, et al. Removal of inflammatory ascites is associated with dynamic modification of local and systemic inflammation along with prevention of acute lung injury: in vivo and in silico studies. Shock. 2014;41:317–23.
111. Kubiak BD, Albert SP, Gatto LA, Snyder KP, Maier KG, Vieau CJ, et al. Peritoneal negative pressure therapy prevents multiple organ injury in a chronic porcine sepsis and ischemia/reperfusion model. Shock. 2010;34:525–34.
112. Cheatham ML, Demetriades D, Fabian TC, Kaplan MJ, Miles WS, Schreiber MA, et al. Prospective study examining clinical outcomes associated with a negative pressure wound therapy system and Barker's vacuum packing technique. World J Surg. 2013;37:2018–30.
113. Kirkpatrick AW, Roberts DJ, Faris PD, Ball CG, Kubes P, Tiruta C, et al. Active negative pressure peritoneal therapy after abbreviated laparotomy: the intraperitoneal vacuum randomized controlled trial. Ann Surg. 2015;262:38–46.
114. Wang J, Kubes P. A reservoir of mature cavity macrophages that can rapidly invade visceral organs to affect tissue repair. Cell. 2016;165:668–78.

115. Demetriades D, Salim A. Management of the open abdomen. Surg Clin North Am. 2014;94:131–53.
116. Regner JL, Kobayashi L, Coimbra R. Surgical strategies for management of the open abdomen. World J Surg. 2012;36:497–510.
117. Godat L, Kobayashi L, Costantini T, Coimbra R. Abdominal damage control surgery and reconstruction: world society of emergency surgery position paper. World J Emerg Surg. 2013;8
118. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–26.
119. Paul JS, Ridolfi TJ. A case study in intra-abdominal sepsis. Surg Clin North Am. 2012;92:1661–77.
120. Lambertz A, Mihatsch C, Röth A, Kalverkamp S, Eickhoff R, Neumann UP, et al. Fascial closure after open abdomen: initial indication and early revisions are decisive factors—a retrospective cohort study. Int J Surg. 2015;13:12–6.
121. Rasilainen SK, Juhani MP, Kalevi LA. Microbial colonization of open abdomen in critically ill surgical patients. World J Emerg Surg. 2015;10:25.
122. Leber GE, Garb JL, Alexander AI, Reed WP. Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg. 1998;133:378–82.
123. Mathes SJ, Steinwald PM, Foster RD, Hoffman WY, Anthony JP. Complex abdominal wall reconstruction: a comparison of flap and mesh closure. Ann Surg. 2000;232:586–96.
124. Choi JJ, Palaniappa NC, Dallas KB, Rudich TB, Colon MJ, Divino CM. Use of mesh during ventral hernia repair in clean-contaminated and contaminated cases: outcomes of 33,832 cases. Ann Surg. 2012;255:176–80.
125. de Vries Reilingh TS, van Goor H, Charbon JA, Rosman C, Hesselink EJ, van der Wilt GJ, et al. Repair of giant midline abdominal wall hernias: "components separation technique" versus prosthetic repair : interim analysis of a randomized controlled trial. World J Surg Springer. 2007;31: 756–63.
126. Yegiyants S, Tam M, Lee DJ, Abbas MA. Outcome of components separation for contaminated complex abdominal wall defects. Hernia. 2012;16:41–5.
127. Rasilainen SK, Mentula PJ, Leppäniemi AK. Components separation technique is feasible for assisting delayed primary fascial closure of open abdomen. Scand J Surg. 2016;105:17–21.
128. Sharrock AE, Barker T, Yuen HM, Rickard R, Tai N. Management and closure of the open abdomen after damage control laparotomy for trauma. A systematic review and meta-analysis. Injury. Elsevier Ltd. 2015; 47:296–306.
129. Atema JJ, de Vries FEE, Boermeester MA. Systematic review and meta-analysis of the repair of potentially contaminated and contaminated abdominal wall defects. Am J Surg Elsevier Inc. 2016;212:982–95.
130. Dinsmore RC, Calton WC, Harvey SB, Blaney MW. Prevention of adhesions to polypropylene mesh in a traumatized bowel model. J Am Coll Surg. 2000;191:131–6.
131. van 't Riet M, de Vos van Steenwijk PJ, Bonthuis F, Marquet RL, Steyerberg EW, Jeekel J, et al. Prevention of adhesion to prosthetic mesh: comparison of different barriers using an incisional hernia model. Ann Surg. 2003;237:123–8.
132. Konstantinovic ML, Lagae P, Zheng F, Verbeken EK, De Ridder D, Deprest JA. Comparison of host response to polypropylene and non-cross-linked porcine small intestine serosal-derived collagen implants in a rat model. BJOG An Int J Obstet Gynaecol. 2005;112:1554–60.
133. Fansler RF, Taheri P, Cullinane C, Sabates B, Flint LM. Polypropylene mesh closure of the complicated abdominal wound. Am J Surg. 1995;170:15–8.
134. Voyles CR, Richardson JD, Bland KI, Tobin GR, Flint LM, Polk HC. Emergency abdominal wall reconstruction with polypropylene mesh: short-term benefits versus long-term complications. Ann Surg. 1981;194:219–23.
135. Brown GL, Richardson JD, Malangoni MA, Tobin GR, Ackerman D, Polk HC. Comparison of prosthetic materials for abdominal wall reconstruction in the presence of contamination and infection. Ann Surg. 1985;201:705–11.
136. Sartelli M, Coccolini F, van Ramshorst GH, Campanelli G, Mandalà V, Ansaloni L, et al. WSES guidelines for emergency repair of complicated abdominal wall hernias. World J Emerg Surg. 2013;8:50.
137. Cornwell KG, Landsman A, James KS. Extracellular matrix biomaterials for soft tissue repair. Clin Podiatr Med Surg. 2009;26:507–23.
138. Caviggioli F, Klinger FM, Lisa A, Maione L, Forcellini D, Vinci V, et al. Matching biological mesh and negative pressure wound therapy in reconstructing an open abdomen defect. Case Rep Med. Hindawi Publishing Corporation; 2014;2014:235930.
139. Badylak SF. Xenogeneic extracellular matrix as a scaffold for tissue reconstruction. Transpl Immunol. 2004;12:367–77.
140. Winters JC. InteXen tissue processing and laboratory study. Int Urogynecol J Pelvic Floor Dysfunct. 2006;17
141. Petter-Puchner AH, Dietz UA. Biological implants in abdominal wall repair. Br J Surg. 2013;100:987–8.
142. Montori G, Coccolini F, Manfredi R, Ceresoli M, Campanati L, Magnone S, et al. One year experience of swine dermal non-crosslinked collagen prostheses for abdominal wall repairs in elective and emergency surgery. World J Emerg Surg. 2015;10:28–35.
143. Primus FE, Harris HW. A critical review of biologic mesh use in ventral hernia repairs under contaminated conditions. Hernia. 2013;17:21–30.
144. Gurrado A, Franco IF, Lissidini G, Greco G, De Fazio M, Pasculli A, et al. Impact of pericardium bovine patch (Tutomesh®) on incisional hernia treatment in contaminated or potentially contaminated fields: retrospective comparative study. Hernia. 2015;19:259–66.
145. de Moya MA, Dunham M, Inaba K, Bahouth H, Alam HB, Sultan B, et al. Long-term outcome of acellular dermal matrix when used for large traumatic open abdomen. J Trauma Inj Infect Crit Care. 2008;65:349–53.
146. Ginting N, Tremblay L, Kortbeek JB. Surgisis® in the management of the complex abdominal wall in trauma: a case series and review of the literature. Injury. 2010;41:970–3.
147. Patton JH, Berry S, Kralovich KA. Use of human acellular dermal matrix in complex and contaminated abdominal wall reconstructions. Am J Surg. 2007;193:360–3.
148. Maurice SM, Skeete DA. Use of human acellular dermal matrix for abdominal wall reconstructions. Am J Surg. 2009;197:35–42.
149. Lin HJ, Spoerke N, Deveney C, Martindale R. Reconstruction of complex abdominal wall hernias using acellular human dermal matrix: a single institution experience. Am J Surg. 2009;197:599–603.
150. Diaz JJ, Conquest AM, Ferzoco SJ, Vargo D, Miller P, Wu Y-C, et al. Multi-institutional experience using human acellular dermal matrix for ventral hernia repair in a compromised surgical field. Arch Surg. 2009; 144:209–15.
151. Lee EI, Chike-Obi CJ, Gonzalez P, Garza R, Leong M, Subramanian A, et al. Abdominal wall repair using human acellular dermal matrix: a follow-up study. Am J Surg. 2009;198:650–7.
152. Pomahac B, Aflaki P. Use of a non-cross-linked porcine dermal scaffold in abdominal wall reconstruction. Am J Surg. Elsevier Inc; 2010;199:22–7.
153. Abdelfatah MM, Rostambeigi N, Podgaetz E, Sarr MG. Long-term outcomes (>5-year follow-up) with porcine acellular dermal matrix (Permacol) in incisional hernias at risk for infection. Hernia. 2015;19:135–40.
154. Booth JH, Garvey PB, Baumann DP, Selber JC, Nguyen AT, Clemens MW, et al. Primary fascial closure with mesh reinforcement is superior to bridged mesh repair for abdominal wall reconstruction. J Am Coll Surg. 2013;217:999–1009.
155. Chand B, Indeck M, Needleman B, Finnegan M, Van Sickle KR, Ystgaard B, et al. A retrospective study evaluating the use of Permacol™ surgical implant in incisional and ventral hernia repair. Int J Surg Elsevier Ltd. 2014;12:296–303.
156. Holihan JL, Nguyen DH, Nguyen MT, Mo J, Kao LS, Liang MK. Mesh location in open ventral hernia repair: a systematic review and network meta-analysis. World J Surg. 2016;40:89–99.
157. Eriksson A, Rosenberg J, Bisgaard T. Surgical treatment for giant incisional hernia: a qualitative systematic review. Hernia. 2014;18:31–8.
158. Bradley MJ, Dubose JJ, Scalea TM, Holcomb JB, Shrestha B, Okoye O, et al. Independent predictors of enteric fistula and abdominal sepsis after damage control laparotomy: results from the prospective AAST Open Abdomen registry. JAMA Surg. 2013;148:947–54.
159. Teixeira PGR, Inaba K, Dubose J, Salim A, Brown C, Rhee P, et al. Enterocutaneous fistula complicating trauma laparotomy: a major resource burden. Am Surg. 2009;75:30–2.
160. Martinez JL, Luque-De-Leon E, Mier J, Blanco-Benavides R, Robledo F. Systematic management of postoperative enterocutaneous fistulas: factors related to outcomes. World J Surg. 2008;32:436–43.
161. Tavusbay C, Genc H, Cin N, Kar H, Kamer E, Atahan K, et al. Use of a vacuum-assisted closure system for the management of enteroatmospheric fistulae. Surg Today. Springer Japan; 2015;45:1102–1111.
162. D'Hondt M, Devriendt D, Van Rooy F, Vansteenkiste F, D'Hoore A, Penninckx F, et al. Treatment of small-bowel fistulae in the open abdomen with topical negative-pressure therapy. Am J Surg. Elsevier Inc; 2011;202:e20–4.

163. Marinis A, Gkiokas G, Argyra E, Fragulidis G, Polymeneas G, Voros D. "Enteroatmospheric fistulae"-gastrointestinal openings in the open abdomen: a review and recent proposal of a surgical technique. Scand J Surg. 2013;102:61–8.
164. Schecter WP, Ivatury RR, Rotondo MF, Hirshberg A. Open abdomen after trauma and abdominal sepsis: a strategy for management. J Am Coll Surg. 2006;203:390–6.
165. Carlson GL, Patrick H, Amin AI, McPherson G, MacLennan G, Afolabi E, et al. Management of the open abdomen. Ann Surg. 2013;257:1154–9.
166. Schein M, Decker GAG. Postoperative external alimentary tract fistulas. Am J Surg. Elsevier; 1991;161:435–438.
167. Schecter WP, Hirshberg A, Chang DS, Harris HW, Napolitano LM, Wexner SD, et al. Enteric fistulas: principles of management. J Am Coll Surg. Elsevier Inc; 2009;209:484–91.
168. Björck M, Kirkpatrick AW, Cheatham M, Kaplan M, Leppäniemi A, de Waele JJ. Amended classification of the open abdomen. Scand J Surg. 2016;105:5–10.
169. Di Saverio S, Tarasconi A, Inaba K, Navsaria P, Coccolini F, Costa Navarro D, et al. Open abdomen with concomitant enteroatmospheric fistula: attempt to rationalize the approach to a surgical nightmare and proposal of a clinical algorithm. J Am Coll Surg. 2015;220:e23–33.
170. Di Saverio S, Tarasconi A, Walczak DA, Cirocchi R, Mandrioli M, Birindelli A, et al. Classification, prevention and management of entero-atmospheric fistula: a state-of-the-art review. Langenbeck's Arch Surg. 2016;401:1–13.
171. Polk TM, Schwab CW. Metabolic and nutritional support of the enterocutaneous fistula patient: a three-phase approach. World J Surg. 2012;36:524–33.
172. Moore FA, Feliciano D V, Andrassy RJ, McArdle AH, Booth F V, Morgenstein-Wagner TB, et al. Early enteral feeding, compared with parenteral, reduces postoperative septic complications. The results of a meta-analysis. Ann Surg. Lippincott, Williams, and Wilkins; 1992;216:172–83.
173. Dissanaike S, Pham T, Shalhub S, Warner K, Hennessy L, Moore EE, et al. Effect of immediate enteral feeding on trauma patients with an open abdomen: protection from nosocomial infections. J Am Coll Surg. 2008;207:690–7.
174. Byrnes MC, Reicks P, Irwin E. Early enteral nutrition can be successfully implemented in trauma patients with an "open abdomen.". Am J Surg. 2010;199:359–63.
175. Reinisch A, Liese J, Woeste G, Bechstein W, Habbe N. A retrospective, observational study of enteral nutrition in patients with enteroatmospheric fistulas. Ostomy Wound Manage. 2016;62:36–47.
176. Yin J, Wang J, Yao D, Zhang S, Mao Q, Kong W, et al. Is it feasible to implement enteral nutrition in patients with enteroatmospheric fistulae? A single-center experience. Nutr Clin Pract. 2014;29:656–661.
177. Nubiola P, Badia JM, Martinez-Rodenas F, Gil MJ, Segura M, Sancho J, et al. Treatment of 27 postoperative enterocutaneous fistulas with the long half-life somatostatin analogue SMS 201-995. Ann Surg. 1989;210:56–8.
178. Lattuada D, Casnici C, Crotta K, Mastrotto C, Franco P, Schmid HA, et al. Inhibitory effect of pasireotide and octreotide on lymphocyte activation. J Neuroimmunol. 2007;182:153–9.
179. Cooper AM, Braatvedt GD, Qamar MI, Brown H, Thomas DM, Halliwell M, et al. Fasting and post-prandial splanchnic blood flow is reduced by a somatostatin analogue (octreotide) in man. Clin Sci. 1991;81:169–75.
180. Navsaria PH, Bunting M, Omoshoro-Jones J, Nicol AJ, Kahn D. Temporary closure of open abdominal wounds by the modified sandwich-vacuum pack technique. Br J Surg. 2003;90:718–22.
181. Al-Khoury G, Kaufman D, Hirshberg A. Improved control of exposed fistula in the open abdomen. J Am Coll Surg. 2008;206:397–8.
182. Layton B, DuBose J, Nichols S, Connaughton J, Jones T, Pratt J. Pacifying the open abdomen with concomitant intestinal fistula: a novel approach. Am J Surg. Elsevier Inc; 2010;199:e48–50.
183. Rekstad LC, Wasmuth HH, Ystgaard B, Stornes T, Seternes A. Topical negative-pressure therapy for small bowel leakage in a frozen abdomen: a technical report. J Trauma Acute Care Surg. 2013;75:487–91.
184. Giner M, Laviano A, Meguid MM, Gleason JR. In 1995 a correlation between malnutrition and poor outcome in critically ill patients still exists. Nutrition. 1996;12:23–9.
185. Cheatham ML, Safcsak K, Brzezinski SJ, Lube MW. Nitrogen balance, protein loss, and the open abdomen. Crit Care Med. 2007;35:127–31.
186. Collier B, Guillamondegui O, Cotton B, Donahue R, Conrad A, Groh K, et al. Feeding the open abdomen. JPEN J Parenter Enteral Nutr. 2007;31:410–5.
187. Cothren CC, Moore EE, Ciesla DJ, Johnson JL, Moore JB, Haenel JB, et al. Postinjury abdominal compartment syndrome does not preclude early enteral feeding after definitive closure. Am J Surg. 2004;188:653–8.
188. Marik PE, Zaloga GP. Meta-analysis of parenteral nutrition versus enteral nutrition in patients with acute pancreatitis. BMJ. 2004;328:1407–10.
189. McClave SA, Heyland DK. The physiologic response and associated clinical benefits from provision of early enteral nutrition. Nutr Clin Pract. 2009;24:305–15.
190. Panel OAA, Campbell A, Chang M, Fabian T, Franz M, Kaplan M, et al. Management of the open abdomen: from initial operation to definitive closure. Am Surg. 2009;75:S1–22.
191. Engels PT, Beckett AN, Rubenfeld GD, Kreder H, Finkelstein JA, da Costa L, et al. Physical rehabilitation of the critically ill trauma patient in the ICU. Crit Care Med. 2013;41:1790–801.
192. Truong A, Fan E, Brower R, Needham D. Bench-to-bedside review: mobilizing patients in the intensive care unit—from pathophysiology to clinical trials. Crit Care. 2009;13:216.
193. Pavy-Le Traon A, Heer M, Narici MV, Rittweger J, Vernikos J. From space to Earth: advances in human physiology from 20 years of bed rest studies (1986-2006). Eur J Appl Physiol. 2007;101:143–94.
194. Herridge MS. Building consensus on ICU-acquired weakness. Intensive Care Med. 2009;35:1–3.
195. Cuthbertson BH, Roughton S, Jenkinson D, Maclennan G, Vale L. Quality of life in the five years after intensive care: a cohort study. Crit Care. 2010;14:R6.
196. Burtin C, Clerckx B, Robbeets C, Ferdinande P, Langer D, Troosters T, et al. Early exercise in critically ill patients enhances short-term functional recovery. Crit Care Med. 2009;37:2499–505.
197. Hodgson CL, Berney S, Harrold M, Saxena M. Clinical review: early patient mobilization in the ICU. Crit Care. 2013;17:207.